the neural tube and is present in structures such as endothelial lining of blood vessels, ganglia and inter-somitic furrows as reported by others.

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Program/Abstract # 153
Temporal analysis of FAK, Src, and ERK1/2 signaling during rapid brain growth of the chick embryo
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Rapid brain growth in the chick embryo begins when a closed, fluid-filled system that generates a positive pressure is established within the hollow embryonic brain. The mechanical strain induced by the pressure of fluid on the intraluminal neuroepithelial surface is essential for normal brain growth. When this pressure is reduced, cell proliferation decreases. Conversely, when this pressure is increased, cell proliferation increases. The pressure of cerebrospinal fluid along the luminal surface of the developing brain is transduced into chemical signals that stimulate cell proliferation. In other cases of pressure-induced proliferation, activation of the same three intracellular signaling proteins, FAK, Src, and ERK1/2, has been shown to up-regulate. FAK is a membrane-associated protein tyrosine kinase that activates a mitogenic signaling pathway in response to mechanical strain. This study assessed the FAK, Src, ERK1/2 signaling pathway in embryonic chick neuroepithelium at five distinct developmental stages. Three pooled samples of de-occulated midbrains and forebrains from chick embryos were collected. Total protein in each sample was determined to assure equal loading of gels for Western blot analysis. Densitometric analysis was used to quantify the temporal pattern of the activated proteins. All samples showed activation of FAK, Src, and ERK1/2 in the neuroepithelium. These data suggest that the mechanically-induced cell proliferation occurring during early embryonic brain development works through the FAK, Src, ERK1/2 pathway.

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Program/Abstract # 154
Essential roles of fibronectin in the development of the left–right embryonic body plan
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Studies in *Xenopus laevis* suggested that cell–extracellular matrix (ECM) interactions regulate the development of the left–right axis of asymmetry; however, the identities of ECM components and their receptors important for this process have remained unknown. We discovered that FN is required for the establishment of the asymmetric gene expression pattern in early mouse embryos by regulating morphogenesis of the node, while cellular fates of the nodal cells, canonical Wnt and Shh signaling within the node were not perturbed by the absence of FN. FN is also required for the expression of Lefty 1/2 and activation of SMADs 2 and 3 at the floor plate, while cell fate specification of the notochord and the floor plate, as well as signaling within and between these two embryonic organizing centers remained intact in FN-null mutants. Furthermore, our experiments indicate that a major cell surface receptor for FN, integrin $\alpha5\beta1$, is also required for the development of the left–right asymmetry, and that this requirement is evolutionarily conserved in fish and mice. Taken together, our studies demonstrate the requisite role for a structural ECM protein and its integrin receptor in the development of the left–right axis of asymmetry in vertebrates.

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Program/Abstract # 155
Region-specific cell shape changes drive morphogenesis of the ciliated organ of asymmetry in zebrafish
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In several vertebrate embryos, motile cilia located in a transient 'organ of asymmetry' create an asymmetric fluid flow that is required for normal left–right (LR) patterning of the embryo. However, how these cilia generate coordinated flow and LR signals is poorly understood. In the zebrafish organ of asymmetry – called Kupffer's vesicle (KV) – cilia are asymmetrically positioned along the anteroposterior (AP) axis with more cilia packed into the anterior region to produce strong leftward flow across the anterior pole. Time-lapse imaging of transgenic embryos expressing GFP in KV cells revealed region-specific cell shape changes during KV development. In wild type embryos, all KV cells initially displayed a similar morphology. However, as development proceeded, cells in the anterior region of KV became thin and elongated to facilitate tight packing, whereas posterior cells became wide and flattened. We refer to this transition as 'KV remodeling.' Importantly, we have found that KV remodeling correlates with the onset of asymmetric flow. To determine whether actomyosin contractility is involved in these cell shape changes, we treated embryos with blebbistatin to inhibit myosin II activity. Blebbistatin disrupted cell shape changes and cilia positioning during KV remodeling, but had no effect on cilium length, number or motility. Disruption of KV remodeling inhibited asymmetric fluid flow in KV and altered LR patterning of the embryo. In addition, morpholino knockdown of Rho kinase 2b (Rock2b) decreased the level phosphorylated (activated) myosin in KV and disrupted KV remodeling. These results indicate that actomyosin activities control cell shape changes required for normal KV morphogenesis and cilia-driven asymmetric flow.

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Program/Abstract # 156
Zebrafish placenta-specific 8.1 (plac8.1) is required for motile cilia morphogenesis and function
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Motile cilia are microtubule-based cellular structures that generate rhythmic beating motion. Conserved among vertebrates, motile cilia are critical during vertebrate development as well as homeostasis. Moreover, cilia defects cause multiple diseases including situs inversus and polycystic kidney disease. Despite the significance of cilia, our understanding of molecular mechanisms regulating motile cilia remains incomplete. Here, we identified zebrafish Plac8.1, a conserved protein of elusive function, as a novel regulator of motile cilia morphogenesis and function. Zebrafish Plac8.1 is a cysteine-rich